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Thermoregulatory Adaptation in Humans and Its Modifying Factors

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Abstract

Humans have extensive abilities to adjust their thermoregulatory activities when responding to changes in both endogenous (exercise) and exogenous (environmental) conditions and thereby maintain thermal homeostasis. Five phenotypic adaptations may be described: reduced heart rate at a fixed work rate; expanded plasma volume; lower core temperature at an equivalent workload; superior sodium and chloride reabsorption from sweat; and an elevated sweat secretion. Thermal factors such as core and skin temperatures are the primary afferent signals that modulate sweat rate and skin blood flow. However, it is also suggested that various non-thermal factors associated with exercise (central command, muscle mechano- and metabo-reflexes, and baroreflexes) can modify sweat rate and skin blood flow. Thermoregulatory controls of sweating and cutaneous vasodilation during heat stress are also modified by circadian changes in core temperature, as well as secretion of endogenous melatonin. Moreover, high humidity reduces sweat efficiency, and therefore core temperature increases during exercise when humidity is elevated at a fixed temperature. In this case, the ability to maintain dry heat loss at the expense of a reduced capacity of evaporative heat loss is seen as an important contributing factor to thermoregulation in humid environments.

Key words: acclimation, adaptation, circadian rhythm, exercise, humans, skin blood flow, sweating

1. Introduction

Humans are currently facing the social, physiological and economic effects of global warming. Since living in "Harmony within Nature" is very important, it is essential these problems are clearly understood. We have effective physiological thermoregulatory mechanisms to defend homeostasis of body temperature against both changing endogenous (exercise) and exogenous environmental conditions (ambient temperature, humidity, radiation and so on). For investigating "Harmony within Nature," it is important to understand thermoregulatory adaptation in humans. Heat adaptation and endurance training induce adaptive changes of the thermoregulatory system. Furthermore, sweating and skin blood flow responses may be modified by non-thermal factors such as central command, muscle mechano- and metaboreflexes, baroreflexes, baroreceptor loading, changes in body fluids (volume reduction, hyperosmolality) associated with exercise, and by the natural circadian rhythm controlled by the hormone melatonin. Therefore, we have briefly reviewed thermoregulatory adaptations to heat and endurance training and the effects of modifying factors on thermoregulatory responses, by focusing on the influences of four factors: 1) heat adaptation and endurance training, 2) exercise, 3) circadian rhythms, and 4) environmental humidity.

2. The Effects of Endurance Training and Heat Adaptation on Thermoregulatory Responses

2.1 Adaptation in the contemporary world

People live and work within a very broad range of naturally occurring air temperatures (–90ºC to >55ºC). Since survival of unprotected individuals at these extremes is very brief, our capacity to live within such environments is wholly dependent upon our ability to develop protective clothing, portable life-support apparatuses and mobile habitats. Indeed, these behavioural capabilities far exceed our physiological capacity to resist thermal energy transfers, or to generate or dissipate

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body heat. Most people from advanced, contemporary societies now have such broad access to these technological advances, that few rarely encounter skin temperatures outside the thermal comfort zone (~33ºC). Daily use of such technology has largely removed modern man from the stresses of life. As a result, contemporary humans have become increasingly more sedentary, and less able to tolerate the stresses of life.

Thermal adaptation occurs through behavioural, morphological, biochemical and physiological adjustments superimposed upon the genetic, phenotypic and physiological status of the individual, thereby improving the relationship between an organism and its surrounding environment. Six stages of heat adaptation may be identified (Taylor & Cotter, 2006), and since cardiovascular and thermal regulatory mechanisms share some common effectors (vascular smooth muscle) and sensors (baroreceptors), many external stresses elicit similar integrated physiological adaptations. For example, repeated endurance exercise and heat exposures elevate core temperature, and stimulate blood flow redistribution and sweat secretion.

2.2 Endurance training

Habitual endurance training is associated with an array of cardiovascular, pulmonary and metabolic adaptations. However, of principal relevance to temperature regulation are changes that facilitate increased peripheral blood flow, since these support metabolic heat removal without compromising blood pressure regulation. In this regard, research indicates that endurance training increases arterial calibre, improves whole-body vasodilatory function and lowers vasomotor tone (Green *et al.*, 2004, 2008). These changes combine to elevate peripheral vascular conductance. An expansion of the blood volume is often observed following endurance training, with increments in plasma volume facilitating heat loss (Tipton *et al*., 2008). In addition, endurance exercise elevates the body tissue temperatures, and runners will routinely complete marathons with a core temperature >40ºC. Such high temperatures are a function of exercise intensity and duration, and endurance athletes produce enough heat, and on such a regular basis, that they must also adapt to training by improving heat dissipation.

2.3 Heat adaptation

Repeated thermal stress elicits adaptations evident within the neural networks and integrating regulatory systems (controller adaptations), that appear as morphological changes (*e.g*., sweat gland hypertrophy), and may be expressed as adjustments within effector processes (*e.g*., altered vasomotor or sudomotor sensitivities). The most common functional effect of heat acclimation is a change in the effector thresholds, with the mean body temperature for effector activation being shifted downwards (Tipton *et al*., 2008). One can also observe effector adaptations, evident from changes in the gain of the effector response. Thus, for a given thermal stimulus, acclimatised individuals frequently display an increased effector sensitivity, such as a greater sweating response for an equivalent change in body temperature. Five phenotypic adaptations may be described: reduced heart rate at a fixed work rate; expanded plasma volume; lower core temperature at an equivalent workload; superior sodium and chloride reabsorption from sweat; and an elevated sweat secretion. However, whilst perhaps the most commonly reported, and certainly the most readily recognised change, is the elevated sweating response, it is possible that this represents only a transitional state as one progresses towards becoming more completely heat adapted (Taylor, 2006).

2.4 Challenging old concepts

Four tenets of heat adaptation physiology may be questioned. First, the nature of the forcing function applied determines the level of adaptation achieved. Traditional heat adaptation protocols use constant thermal and metabolic stresses to elicit adaptation. However, superior physiological adaptation can be achieved by gradually increasing the stress applied as adaptation occurs (Taylor & Cotter, 2006). Second, heat adaptation-induced increases in plasma volume are not transitory, but can be sustained if the forcing function is elevated as adaptation progresses (Patterson *et al*., 2004b). Third, heat adaptation is not accompanied by a peripheral redistribution of sweat (Patterson *et al*., 2004a). Finally, it has been considered that a low sweat rate is indicative of an inferior state of heat adaptation. While this may be correct in many cases, it is not a correct interpretation for those who live in tropical climates. For such individuals, the ultimate level of heat adaptation is associated with reduced and very efficient sweat secretion during thermal loading (Taylor, 2006).

3. Neural Control of Sweating and Skin Blood Flow during Exercise

3.1 Factors associated with exercise that modify thermoregulatory responses

Thermal factors such as core and skin temperatures are the primary feedback signals for sweat rate and skin blood flow, and thus these responses are typically expressed as a function of internal or mean body temperatures (Nadel *et al.*, 1971). However, van Beaumont and Bullard (1963) first reported that sweating occurred immediately (within 1.5-2 sec) with the onset of dynamic exercise in warmed subjects, and these responses occurred prior to any measurable elevation of core temperature. They concluded that non-thermal factors associated with exercise contributed to this increase. It is suggested that a number of non-thermal factors associated with exercise (central command, muscle mechano- and metabo-reflexes, and baroreflexes) can modify the aforementioned thermoregulatory responses (Fig. 1). In addition, the effect of body fluid regulation on thermoregulatory responses should also be considered.

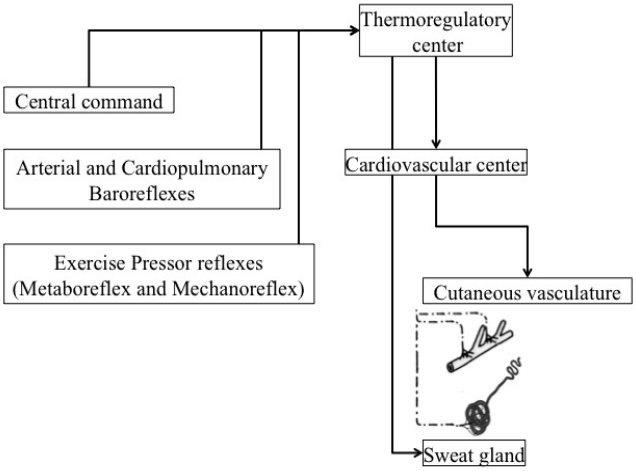

Fig. 1 Effects of non-thermal factors on cutaneous vascular and sweat gland responses.

3.2 Sweating

Shibasaki *et al*. (2003) measured sweat rate during 2 min. of isometric handgrip exercise with and without augmented central command, via administration of a partial neuromuscular blocking agent. After administration of the agent, force generation could not be maintained; however, the sweat rate during this period remained elevated similar to the response to the exercise bout without neuromuscular blockage. These findings provide evidence that central command can modulate sweat rate.

Circulatory occlusion just prior to the end of exercise stimulates muscle metaboreceptors while central command and muscle mechanoreceptor activity are removed. During circulatory arrest after isometric handgrip exercise, the sweat rate remains elevated and then decreases upon release of the ischemic challenge (Crandall *et al.*, 1998; Kondo *et al.*, 1999; Shibasaki *et al.*, 2001) without any effect of baroreceptors on sweating (Shibasaki *et al*., 2003). Furthermore sweating is augmented during dynamic exercise with leg compression (Kacin *et al.*, 2005). Together these findings suggest that muscle metaboreceptor stimulation is capable of modulating the sweat rate during both isometric and dynamic exercise.

Passive cycling or limb movement is one of several methods used to selectively stimulate muscle mechanoreceptors. Kondo *et al*. (1997) adopted passive limb movement in subjects exposed to a warm environment, which caused slight elevations in the sweat rate at the onset of limb movement. In addition, Shibasaki *et al*. (2004) had subjects perform passive cycling after moderate exercise. During the recovery following moderate exercise, the sweat rate was greater when the subjects' legs were passively cycled relative to when the subjects rested. These studies demonstrated that muscle mechanoreceptors are capable of modulating sweat rate during exercise.

Finally, hypovolemia reduced the slope of the relationship between the increase in core temperature relative to the increase in sweating, but did not change the core temperature threshold for the onset of sweating (Nadel *et al.*, 1980; Fortney *et al.*, 1981). Plasma hyperosmolality inhibits the core temperature threshold for the onset of sweating but not the aforementioned slope (Takamata *et al.*, 1997).

3.3 Skin blood flow

Friedman *et al*. (1991) found that a reduction in cutaneous vascular conductance (CVC) observed at the onset of dynamic exercise in normothermic subjects was not mediated by central command, in that CVC did not decrease when the subjects tried to perform exercise during partial neuromuscular blockade. In contrast, recent data suggest that during isometric exercise of heat stressed subjects, central command can contribute to the reduction in CVC observed during this form of exercise (Shibasaki *et al.*, 2005), and is primarily due to withdrawal of cutaneous active vasodilator activity.

During a period of post-isometric-exercise ischemia of heat stressed subjects, Crandall *et al*. (1998) found that CVC remained reduced, and upon release of ischemia returned to pre-exercise levels. They concluded that the reduction in CVC during isometric exercise in heat stressed subjects can be mediated by muscle metaboreceptor stimulation resulting from withdraw of cutaneous active vasodilation (Crandall *et al.*, 1995, 1998).

Shibasaki *et al*. (2004) showed that CVC response during semi-recumbent passive cycling (*i.e*., primarily muscle mechanoreceptor stimulation) after moderate exercise was similar to that in subjects not cycling during recovery. This result indicated that stimulation of muscle mechanoreceptors does not modulate CVC

Johnson and Park (1981) showed that upright posture and exercise each contributed to an increased threshold for cutaneous vasodilation. Moreover, numerous observations show that baroreceptor unloading due to lower-body negative pressure and head-up tilt in heat stressed subjects is capable of reducing cutaneous vasodilator activity. Hypovolemia also reduced the slope of the relationship between the increase in cutaneous vasodilation and the increase in core temperature (Nadel *et al.*, 1980). When core temperature approaches ~38°C during dynamic exercise, the rate of increase in CVC is substantially attenuated (Brengelmann *et al.*, 1977). The feedback mechanism to maintain cardiac filling pressure may be the mechanism resulting in an attenuated rise in CVC when core temperature is substantially elevated (Nose *et al.*, 1990). Similar to that observed with sweating, elevated plasma osmolality increases the core temperature threshold for the onset of cutaneous vasodilation (Takamata *et al.*, 1997).

4. Modification of Circadian Variation in Human Thermoregulatory Responses

4.1 Circadian variation of sweating and skin blood flow responses during heat stress

It is well recognized that the circadian rhythm in resting core temperature in humans shows a nadir in the early morning and a peak in the evening. This rhythm in

core temperature results from an endogenous circadian rhythm in heat production and heat dissipation (Aschoff & Heise, 1972). A resting individual is in a heat-gain mode in the morning and in a heat-loss mode in the late evening (Waterhouse *et al.*, 2005). Effector responses to thermoregulatory challenges also show diurnal or nocturnal variation during cycling exercise (Wenger *et al.*, 1976; Stephenson *et al.*, 1984). For example, the circadian variation in core temperature thresholds for the onset of sweating and active cutaneous vasodilation during dynamic exercise clearly shifted in parallel to the circadian rhythm in the resting core temperature (Stephenson *et al.*, 1984). However, these variations in thermoregulatory sweating and cutaneous vasodilation during dynamic exercise are modulated by both thermal and non-thermal factors as mentioned in the previous chapter.

Aoki *et al*. (2001) compared the thermoregulatory sweating and cutaneous vasodilation evoked by passive heating in the early morning and evening. The core temperature thresholds for the onset of sweating and active cutaneous vasodilation shifted to higher core temperatures in the evening compared with thresholds observed in the early morning (Figs. 2 and 3). The slope of the relationship between the increase in core temperature relative to the increase in sweating did not differ between the early morning and evening. On the other hand, the slope of the relationship between the increase in core temperature and the increase in cutaneous vasodilation declined in the early morning compared with the slopes observed in the evening.

4.2 Modification of exogenous and endogenous melatonin on thermoregulatory responses

In humans, the nadir in core temperature occurs during the phase from midnight to early morning and skin blood flow in the distal limbs peaks during this phase (Smolander *et al*., 1993) when endogenous melatonin levels rise to their nocturnal maximum (Kräuchi *et al.*, 2006). Time courses suggest a causal relationship between the nocturnal increase in endogenous melatonin and the nocturnal decrease in core temperature. On the other hand, melatonin secretion is typically suppressed by bright light and, thus, endogenous melatonin levels are almost zero during daytime.

An oral dose of melatonin (3 mg) administered in the daytime induced a decrease in resting core temperature. Also, core temperature thresholds for the onset of sweating and active cutaneous vasodilation during passive heating in the evening were shifted to lower core temperatures by exogenous melatonin (Figs. 2 and 3) (Aoki *et al.*, 2006). Moreover, exogenous melatonin administration reduced the slope of the relationship between the increase in core temperature and the increase in cutaneous vasodilation, but did not change the slope of the relationship between the increase in core temperature relative to the increase in sweating.

It is well known that the circadian rhythms in endogenous melatonin and resting core temperature can

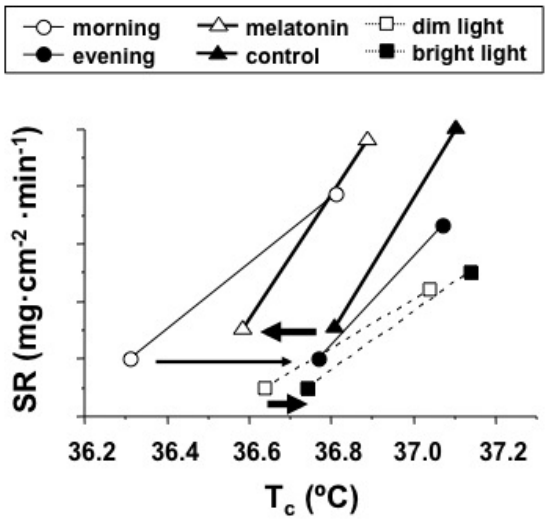

Fig. 2 Sweating response to passive heating from the onset of sweating up to the time of achieving a steady state. SR is shown as a function of T_c. The regression lines are means from all subjects.
SR; sweat rate, T_c; core temperature.

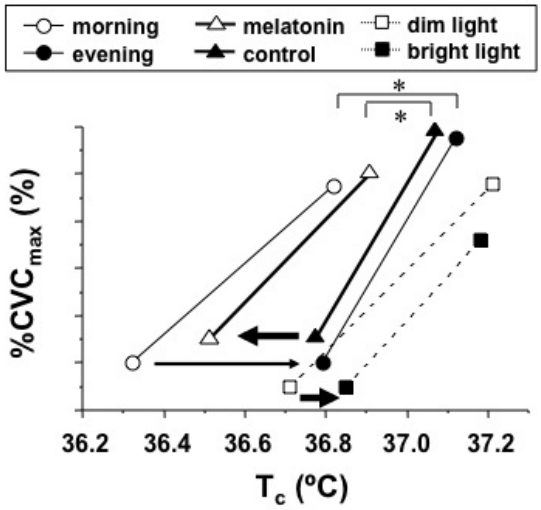

Fig. 3 Cutaneous vasodilator response to passive heating from the beginning of vasodilation up to the time of achieving a steady state. CVC, expressed as a percentage of maximal CVC (%CVC_{max}), is shown as a function of T_c. The regression lines are means from all subjects.
CVC; cutaneous vascular conductance, T_c; core temperature. *; The slope of relationship between the increase in core temperature relative to the increase in cutaneous vasodilation significantly declined ($P<0.05$).

be entrained or phase-shifted by artificial bright light exposure (Czeisler *et al.*, 1989; Strassman *et al.*, 1991). For example, bright light (BL) exposure (>2,000 lx) for a single night suppressed secretion of melatonin and attenuated the fall in core temperature from the night to the early morning (Strassman *et al.*, 1991). Indeed, BL exposure (2,800 lx) at night suppressed melatonin and attenuated the fall in core temperature during the night. Also, the core temperature thresholds for the onset of sweating and active cutaneous vasodilation during

passive heating in the early morning were shifted to higher core temperatures by BL exposure (Figs. 2 and 3) (Aoki *et al.*, 2005). However, there was BL exposure that had no effect on the slope of the relationship between the increase in core temperature relative to the increase in sweating or cutaneous vasodilation.

5. The Effect of Humidity on Thermoregulatory Responses during Prolonged Intense Exercise

5.1 How does humidity affect thermoregulatory responses?

Prolonged exercise in warm to hot ambient temperatures inevitably results in thermoregulatory strain as the body seeks to maintain homeostasis. The addition of highly humid conditions exacerbates physiological strain, a fact that is recognized by the weight given to relative humidity in the wet bulb globe temperature (WBGT) heat stress index.

Perhaps the most comprehensive study examining the influence of relative humidity on the human thermal environment was performed by Winslow and colleagues (1937) who passively exposed their subjects to varying levels of ambient temperature and relative humidity. It was concluded that when environmental temperature was above 25ºC, the relative humidity of the atmosphere would have a detrimental influence upon physiological responses. Since then, there have been various experiments examining the influence of humidity on exercise performance (Pandolf *et al.,* 1974; Nielsen *et al*., 1997; Gonzalez *et al*., 1974; Kobayashi *et al*., 1980; Frye & Kamon, 1983). A common observation from these experiments was that greater thermoregulatory strain was experienced at higher levels of environmental humidity. Compared with lower humidity levels, exercise at high environmental humidity in these studies caused a steady rise in core temperature with no sign of thermal equilibrium and mean skin temperature rising to a higher level. With rising skin temperature at elevated rectal temperature, the ability to transfer heat from the core to the skin is reduced, thus causing an increase in body heat storage which ultimately results in fatigue as core temperature reaches a critical level of around 40ºC (Nielsen *et al*., 1997). This increased thermoregulatory strain during exercise in humid environments is attributed to a limited capacity to evaporate sweat due to the decline in the maximal evaporative capacity of the environment.

A distinct feature of thermoregulatory responses in humid environments is a reduced rate of sweating with increasing core temperature during prolonged exposure (Candas *et al.,* 1983), a phenomenon referred to as hidromeiosis (Brown & Sargeant, 1965). Hidromeiosis refers to a condition associated with increased skin wettedness that leads to a reduction in sweat secretion. However, this phenomenon and the underlying proposed mechanism remain controversial.

In addition to a reduced sweat rate, earlier observations have highlighted a decline in sweating efficiency with increasing level of humidity (Libert *et al*., 1975; Candas *et al.,* 1979). Sweating efficiency, defined as the ratio between evaporative rate and sweat rate, is an important index that evaluates the effect of the sweat rate on thermal regulation. A decline in sweating efficiency indicates that sweat produced during exercise in a humid environment is rendered relatively ineffective in contributing to heat dissipation as it merely drips from the skin surface, resulting in an increased area of skin covered with sweat (skin wettedness).

5.2 Effect of humidity in earlier studies

The current understanding of the physiological responses during prolonged exercise in humid environments has been limited to low ($\leq \dot{V}O_2$ 25% max) and moderate (40% to 50% $\dot{V}O_2$ max) exercise (Pandolf *et al.,* 1974; Nielsen *et al*., 1997; Gonzalez *et al*., 1974; Kobayashi *et al*., 1980) involving sedentary to moderately trained subjects. Highly trained athletes have been shown to have generally superior capability to cope with heat stress due to their higher sweat rates and higher skin blood flow rates (Tipton *et al.*, 2008).

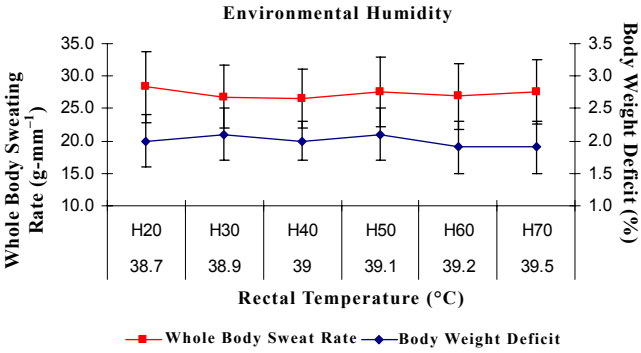

Fig. 4 Whole body sweat rate (WBSR) at the end of 60 minutes exercise across different levels of relative humidity (n=11). WBSR was not reduced with rising humidity in highly trained men.

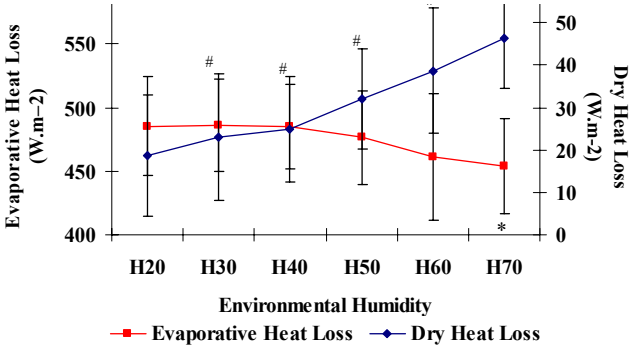

Fig. 5 Evaporative and dry heat loss values at 60 minutes of exercise under increasing level of environmental humidity, relative humidities from 20% (H20) to 70% (H70): n=10. Evaporative heat loss decreased gradually while dry heat loss was increased with rising humidity. # indicates significant difference from the H20 environmental condition (P<0.05).

In a study examining the systematic changes in humidity involving a group of highly trained, male distance runners (mean $\dot{V}O_2$ max = 62.9 ml/kg/min), Che Muhamed and colleagues found that subjects were able to complete a 60 minutes intense running (70% $\dot{V}O_2$ max) exercise despite experiencing thermoregulatory stress. While earlier studies have shown a decline in sweating during prolonged exercise in humid environments, highly trained endurance athletes were able to maintain sweating rate across varying levels of relative humidity as demonstrated in Fig. 4 (Che Muhamed *et al*., unpublished data). In addition to the ability to maintain sweat rate in highly trained subjects during prolonged exercise, the ability to maintain dry heat loss at the expense of a reduced evaporative heat loss capacity is seen as an important contributing factor to thermoregulation in humid environments (Fig. 5).

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